

COVID-19, myocarditis, and the other side of the bed

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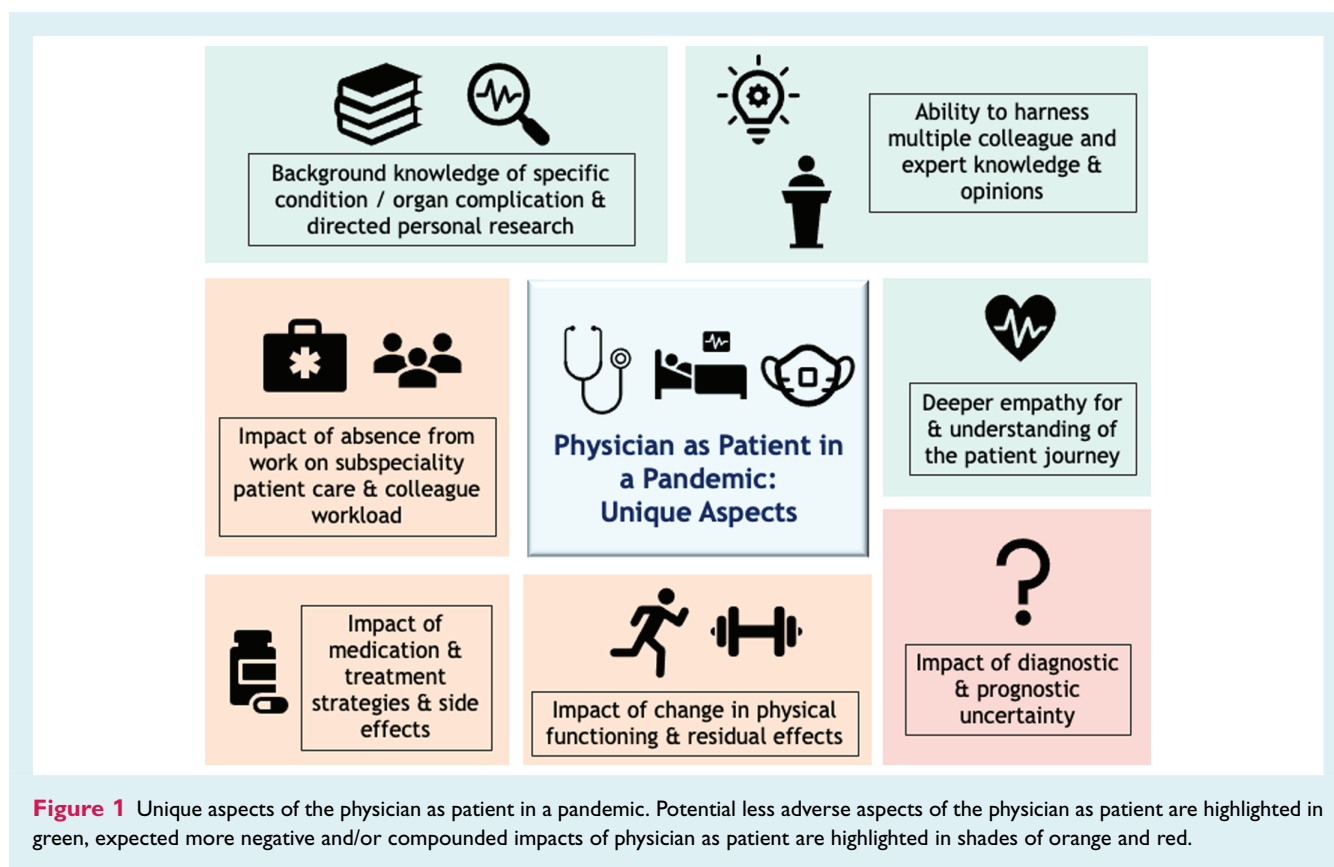
On the 15th of September, 2019 I had the honour of presenting in a session entitled 'How to diagnose and manage inflammatory cardiomyopathies' at the Heart Failure Society of America Annual Scientific Meeting held in Philadelphia, USA, where my particular brief included discussing treatment strategies for fulminant myocarditis. It was an exciting, thought-provoking and absorbing session (of course I am biased), highlighting current diagnostic and treatment paradigms for acute myocarditis and inflammatory cardiomyopathies alongside future avenues of clinical and research importance associated with this frequently chameleonic group of conditions. Little did we know then that much less than a year later, a whole spectrum of newly proposed inflammatory cardiac syndromes,¹ triggered by one specific viral pathogen, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), would have hijacked the clinical landscape of our acute cardiac, intensive care and cardiomyopathy/heart failure services. Little did I know then that exactly 1 year later, I myself would still be recovering from SARS-CoV-2 myocarditis.

The tale of the overall healthy, underlying-condition-less, pre-morbidly highly active 'younger' healthcare professional contracting SARS-CoV-2 and suffering physiologically pervasive consequences at odds with the designated 'mild' label has been told before. I'm grateful now that I had no idea what lay in store on the day I was confirmed COVID-19 positive; at the time I accepted it as a necessary inevitability to be able to move forward with clinical certainty after the initial relief of a false negative swab the week before was obliterated by refractory, daily viremic assault. Each time I recount the story I find myself adding the phrase '... and I had ran 50 miles the week before...', an almost involuntary verbal tic in a desperate attempt to retain some semblance of a proud former identity – one which has been lost in a blur of 'good and bad days' and a new baseline exertional limitation that belies a priori knowledge of such a fit state. But where does COVID-19 end and myocarditis begin? Was myocardial involvement to my illness an upfront co-invaded passenger since the early infectious phase or a later (weeks) phase inflammatory response by an over-reacting host (me)? Surely as a subspecialist in this very field, I am especially equipped to differentiate symptom initiation and/or evolution that

might shed more light – at least anecdotally – on one of the (many) conundrums associated with this diagnosis? Unfortunately, this did not bear out. Since my predominantly systemic syndrome first began, chest pains of various features and flavors had been daily bedfellows and in my boundless experience, I wrote them all off as benign actors. However, I do recall the moment I accepted the veracity of those underlying nagging thoughts questioning this assessment. The pain at this particular moment of clarity was exertional, limiting, central, felt like a pressure and was associated with a feeling that if not quite 'angor animi' was certainly close.

If myocarditis is and has always been a chameleon of cardiology, then SARS-CoV-2 myocarditis may go on to be its pinnacle. As of course expected with a condition in its infancy, many known unknowns and unknown unknowns remain to be discovered, and despite identifying as an 'expert' in the myocardium and its whims, prejudices and vulnerabilities, and subsequently being held hostage at its hands, I feel as if I know less now than before this all started. I was diagnosed approximately 1 month after the initial SARS-CoV-2 diagnosis, several days after the malignant chest pain episode described above, and 5 weeks after general viral symptom onset. Cardiac magnetic resonance imaging per standardized myocarditis criteria played a leading role in my diagnosis, with good supporting performances by traditional studies including echo- and electro-cardiography. Mine was a 'bi-layer' carditis, with evidence also of pericardial involvement. Van Linthout *et al.*¹ in a well-written and expansive editorial outlined 11 single cases of 'probable' SARS-CoV-2-associated myocarditis, highlighting a rather heterogeneous group (albeit all nasopharyngeal swab positive for the virus) with variable clinical presentations and pathways to diagnosis. Remarkably, in one of those cases published earlier in this Journal,² viral particles looking highly suspicious for the now-ubiquitous morphology of SARS-CoV-2 were demonstrated via endomyocardial biopsy in a critically ill patient with a combination of acute respiratory distress syndrome and cardiogenic shock. I did not undergo cardiac biopsy given the preponderance of the left ventricular free wall in my syndrome, and of course thankfully, the lack of haemodynamic compromise or malignant arrhythmia. It is clear that the remarkable heterogeneity in clinical cardiac syndromes associ-

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ated with this disease highlights the likelihood for multiple potential mechanisms underscoring SARS-CoV-2-mediated myocardial injury.^{3,4} For healthcare systems taking care of these patients, focused research and investigation with the aim of distinguishing these subtypes of SARS-CoV-2 cardiac pathology (direct and indirect) are urgently needed to further clinical understanding and facilitate targeted therapies.^{1,5}

But the primary goal of this article is neither illustrative case report nor detailed topic review, as well-trodden and infinitely more comfortable as those shoes are to wear. Rather, the brief is to present a birds-eye view of the physician as patient, the sub-specialist as sub-specialist condition sufferer, the one on the far side of the bed as the one in the bed (Figure 1). All of this through the looking glass of a pandemic, and its already heavy burdens on work and home life (and everything in between). What particular insights wearing these hats can I offer? Firstly, from a patient perspective, it is OK for their doctor not to have all the answers. There are no sub-specialists in SARS-CoV-2 myocarditis to call (yet) but I still had the privilege of being able to reach out to many mentors, current and former colleagues, and bona-fide experts in the fields of cardiology, heart failure/transplant, cardiac imaging, immunology and infectious diseases, both nationally and internationally, all of whom provided me with many unique pearls of wisdom but more importantly, empathy and reassurance. Even though we might not have all the answers, or even yet all the questions, traditional biomarkers of prognosis appear no less relevant. One of the most difficult aspects was trying to keep up with what

seemed like an almost constant barrage of information coming from all angles – not just the usual evidence-based resources and relevant society and academic websites, but also twitter, other social media, daily broadsheets and news bulletins. It was surreal being in the epicenter of the information storm, being bombarded with supposed theories and aetiologies in real time, and at moments rather than diving deeper into it given my career bias and personal diagnosis, my instinct was to run as far away as I could (sadly, speaking figuratively). Hence, my immense gratitude for the personnel both on the ground and on the phone who were willing to take that burden off my shoulders and then some. Again, patients do not need physicians to know it all, but they do need them to care enough to reach out, call, debate and discuss, on a backdrop of clinical accountability, empathy and respect.

As the weeks and then months rolled by, further insights have revealed themselves. As important as the afore-mentioned prognostic markers are, they do not tell the whole story. I now realize those times that I have walked into clinic rooms borderline euphoric – often in the case of having reviewed that patients' repeat echocardiogram prior and witnessed a wonderfully improved ejection fraction – and subsequently expected the patient to have the same reaction was naïve and in some ways unfair. One test does not a person define, and continuing the metaphor of ejection fraction, while infinitely preferable to have a normal one, it does not mean that that same patient does not have some distance to go physically and/or mentally towards overcoming their condition. Similarly, just because it has now 'all worked out' and

the desired endpoint or positive resolution has been reached, does not negate the negative experience, or in some cases, the trauma, that our patients have experienced to get to this point. Specific to SARS-CoV-2 anything, the fear of the still completely unknown or 'anybody's guess' longer-term sequelae or consequences is also a relevant factor unique to this cohort. As cardiologists, we are particularly numbers and endgame driven – a by-product of a phenomenally successful evidence-driven half century – but since going through this I have a hunch that taking time to acknowledge and validate patients' experiences no matter the destination will always be highly appreciated.

Finally, a crucial learning point of this experience for me has been its reinforcement of the true privilege associated with my usual position as a clinician. The 'Other Side of the Bed' as I came to call it, no matter the condition, is a fearful, vulnerable and exposed place to be. As doctors and caregivers on our daily bedside rounds, supervising and guiding our patients' journeys through illness, we are the lucky ones. Although in recent years I have been particularly drawn to the academic realm of medicine and its challenges and rewards, I have realized that no publication acceptance email or invited speaker request will ever match the joy of those 'ordinary' clinical moments when things go right, such as when an elderly gentleman can finally get his shoes and socks on after a significant diuresis, or when a younger patient with a very low ejection fraction responds beautifully and progressively to goal-directed medical therapy. Next time though, I promise to keep the euphoria to myself.

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